

Gender, Race, Class and Self-Reported Sexually Transmitted Disease Incidence

By Koray Tanfer, Lisa A. Cubbins and John O.G. Billy

Multivariate analysis of data from two nationally representative surveys of adult men and women indicates that the likelihood of a self-reported sexually transmitted disease (STD) infection varies by gender, race and socioeconomic status, even after accounting for differences in sexual and health care behaviors. Women and black respondents are more than three times as likely to report an STD infection as men and white respondents; men and women with 12 or fewer years of education are about 30% less likely than those with more schooling to report having had an STD. Income, welfare status and access to health care have no significant association with self-reported STD incidence, but sexual behavior is strongly related. Men and women who have engaged in anal intercourse, have paid for sex or have had one-night stands are significantly more likely than those who avoid these behaviors to report an STD. Further, the likelihood of an STD dramatically increases with the lifetime number of sex partners reported: Compared with men and women who have had only one partner, those who report 2–3 partners are five times as likely to have had an STD; the odds are as high as 31:1 for those who report 16 or more partners. (Family Planning Perspectives, 27:196–202, 1995)

Sexually transmitted diseases (STDs) are a major public health problem in the United States, generating societal costs in excess of \$3.5 billion annually.¹ During the last decade, both the number of STDs and their complexity have increased dramatically; more than 50 organisms and syndromes are now recognized. Although many of these diseases have long been known, others have recently achieved prominence because new diagnostic methods have helped investigators to describe their extent, method of transmission and clinical consequences.

STDs affect almost 12 million Americans each year, and nearly 50 million Americans may already have acquired viral STDs, which are not curable and infect the individual for life.² Gonorrhea is the most frequently reported STD; almost 700,000 cases were reported in 1990. Chlamydia is another common sexually transmitted bacterial pathogen; no formal surveillance system exists, but estimates

derived from a variety of sources suggest that 3–4 million persons are infected with chlamydia annually.³

Women bear disproportionate consequences from gonorrhea and chlamydia because of the risk of pelvic inflammatory disease (PID), which often leads to such adverse sequelae as infertility and ectopic pregnancy. Some bacterial or viral STDs may affect infants either in utero or at birth. Additionally, other population subgroups are at increased risk of STDs. STD infection is more prevalent among blacks than among members of other racial groups, and is more common among those of low socioeconomic status than among those of higher status.⁴

Race and socioeconomic status may be different manifestations of the same phenomenon, but this supposition has not been unequivocally demonstrated. In any case, these two characteristics are clearly related to adverse health conditions, including cancer, diabetes and cardiovascular diseases, as well as STDs and AIDS.⁵ Gender, race and social class not only influence risk behaviors, but also are presumed to affect the efficiency of transmission of some STDs, the ease with which infection is detected and care-seeking behaviors.⁶

In this article, we examine gender, race and class differences in the likelihood of ever having had an STD among a national sample of 20–37-year-old women and 20–39-year-old men.

Conceptual Framework

Three possible explanations may account for gender, race and class differentials in the distribution of STDs: differential biological disposition to acquiring certain diseases; differential sexual behaviors that increase the risk of infection; and differentials in preventive health behavior and access to and use of health care services. These explanations can be viewed in the context of a social-behavioral model that is based on the premise that STD acquisition is a function of the probability of exposure to infection, the probability of infection if exposed and the probability of disease if infected.⁷

The probability of exposure to an infected person is a function of both the number of new sex partners per unit of time and whether any given partner is infected. The latter is determined by the prevalence of infection within a community.

The probability that infection will occur after exposure is primarily a function of biological variables, including factors related to the pathogen, the infectiousness of the source and the susceptibility of the host. Behavioral risk factors, such as types of sexual behavior (e.g., anal vs. vaginal intercourse), frequency of sexual contact and preventive health care (e.g., use of condoms), also influence the likelihood of infection following exposure.

The probability that an infection will lead to a disease is influenced chiefly by health care-seeking behavior, which in turn is determined by such factors as the individual's willingness and ability to obtain health care services, as well as the availability, accessibility and cost of health care in the community. Once health care services have been obtained, compliance with therapy largely determines whether the infection will progress to a disease.

The behavioral risk factors are proximate determinants of STD acquisition, and they operate through the three parameters described above (exposure, infection and disease). We focus here on four risk factors related to sexual behavior (lifetime number of vaginal sex partners, engaging in one-night stands, exchanging sex for money or drugs, and engaging in anal intercourse) and two related to health

Koray Tanfer and John O. G. Billy are senior research scientists at Battelle Memorial Institute, Centers for Public Health Research and Evaluation, Seattle. At the time this article was written, Lisa A. Cubbins was research associate at Battelle Memorial Institute; she is currently assistant professor of sociology at the University of Cincinnati. The authors gratefully acknowledge the support for this work through National Institute of Child Health and Human Development (NICHD) grants HD-26288 and HD-26631, and National Institute of Allergy and Infectious Diseases (NIAID) grant AI-34360. The views expressed here do not necessarily reflect the opinions or policies of the Battelle Memorial Institute, the University of Cincinnati, the NICHD or the NIAID.

care behavior (having a regular doctor and having health insurance).

Race has traditionally been tagged a risk marker for both sexual and health care behaviors.⁸ For example, the onset of sexual activity is earlier among young black men and women than it is among whites.⁹ Further, black men have a greater lifetime number of sex partners than men of other races.¹⁰ It is also well documented that minority populations are characterized by poor health education, poor health care-seeking behavior, and poor access to diagnostic and therapeutic health services.¹¹ Additionally, because of the overall higher prevalence of STDs among the black population, and because there is a tendency toward selecting partners of the same race, blacks are at relatively high risk of STDs.

Socioeconomic status is thought to influence sexual behavior—specifically, to raise the likelihood of having multiple partners and engaging in a variety of sexual behaviors with those partners—for two reasons. First, education tends to have a liberalizing influence, promoting a more permissive sexual ideology among higher status men and women.¹² Second, higher status women and men are attractive mates because of their wealth, power and prestige.¹³

On the other hand, men and women of high socioeconomic status also have a greater sense of self-efficacy, better access to health care services and a higher likelihood of having health insurance coverage than those of lower status.¹⁴ These characteristics promote the use of measures to prevent STD infection and of STD-related medical services. Further, men and women of high status tend to have low-risk sex partners because of class homogamy with respect to education, income and social class of origin,¹⁵ and because high socioeconomic status is associated with relatively low STD rates. These factors are hypothesized to mitigate the positive influence of socioeconomic status and produce an overall negative relationship between class and the likelihood of STD infection.

Gender differences in STD rates are partly attributable to differences in the efficiency of transmission of some pathogens and the ease with which infection can be detected.¹⁶ The difference in the efficiency of transmission results partly because the contact with pathogens after sexual exposure is more extended among women than among men. That is, if the male partner has an STD, the infected semen remains in the vagina following intercourse; in contrast, if the female partner is infected, the male's

exposure to the pathogens is limited to the duration of coitus. The cervix may also be more susceptible to infection than the male's urethra.¹⁷

Furthermore, STDs are more difficult to detect in women than in men. For anatomical reasons, certain STDs may go unnoticed in women. Moreover, the large number and variety of cells and bacteria that are normally present in the vaginal vault reduce the sensitivity of certain specimen tests.¹⁸

Additionally, sexual and health care behaviors differ by gender. On average, women become sexually active at a later age than men and have fewer sex partners per unit of time.¹⁹ Patterns of sex partner selection also differ. Men are far more likely than women to engage in casual sex with a date or someone they have just met. A nondiscriminating approach to sex partner selection increases the likelihood of sexual contact with members of high-risk groups and with infected partners.²⁰ Women are more likely to meet their potential sex partners through less-casual associations, and tend to know them better and for a longer period before having sex with them.²¹

In general, women are more likely than men to seek health care.²² However, care for STDs seems to be an important exception to this observation for several reasons. First, STD infections in women are far more likely to be asymptomatic than infections in men.²³ Second, when symptoms do occur, they are generally less obviously attributable to STDs in women than in men.²⁴ Third, seeking health care from an STD clinic may be more stigmatized for women than for men.

A number of other risk markers in addition to race, gender and socioeconomic status are associated with the incidence and prevalence of STDs. Marital status is a consistent predictor of many dimensions of sexual behavior, in that single people tend to engage in riskier sexual practices than married couples.²⁵ Further, married men are less likely than single men to use condoms,²⁶ and married couples are more likely than others to seek medical care.²⁷

Age is another factor. Younger men and women are generally at a higher risk of acquiring an STD than older people because they accumulate sex partners more rapidly, they are less likely to seek health care and they are more likely to select high-risk partners.²⁸

In addition, STD prevalence is relatively high among Hispanics. Therefore, given the tendency toward ethnic homogamy in partner selection, Hispanic men and women are at increased risk of STDs.²⁹

We might also expect religion to be as-

sociated with the risk of STD infection. To the degree that religions encourage restrictive sexual ideologies, adherents would be expected to have few sex partners and to engage in a narrow range of sexual behaviors.³⁰ Moreover, religious homogamy would tend to lead to selection of low-risk partners. This negative effect on STD risk, however, may be offset to the extent that conservative religious ideologies stigmatize STDs, and consequently reduce the likelihood of both preventive behavior and the use of STD clinics and related services.

Data

The data used in this analysis were obtained from the National Survey of Men (NSM)³¹ and the National Survey of Women (NSW),³² both conducted in 1991. The NSM was based on a multistage, stratified, clustered, disproportionate area probability sample of households in the contiguous United States. The sample consisted of 20–39-year-old men. In-person interviews were conducted with 3,321 men, 70% of the eligible sample.

The NSW sample, consisting of 1,669 women aged 20–37, comprises two subsamples, both based on a multistage, stratified, clustered area probability design. Women in the first subsample were originally interviewed in 1983, when they were 20–29 years old and had never been married. This subsample included a sample of college dormitories and sorority houses. The 1,314 respondents (including 165 college students) were traced, and 929 (71%) were reinterviewed in 1991. Interviews for the second subsample were completed with 740 women aged 20–27, regardless of marital status. The response rate for this panel was also 71%. Both subsamples were weighted to account for differential selection probabilities, oversampling and nonresponse.

In both the NSM and the NSW, the black population was oversampled to allow adequate representation in the database. Other population groups known to have an elevated risk of contracting an STD were not oversampled.

The combined sample used in this analysis includes 3,321 men (1,238 blacks and 2,083 members of other racial groups) and 1,669 women (728 blacks and 941 others). These racial groups included 241 Hispanic men and 123 Hispanic women. Distributions of the sample by age and marital status reflect those of the U.S. population at these ages. The data have been weighted to account for the effects of stratification, clustering, disproportionate area sampling and the oversampling of black men and women, as well as for the effects

Table 1. Percentage of U.S. men aged 20–39 and women aged 20–37 who have ever had an STD, by type, according to gender and background variables, 1991

Gender and variable	All*	Chlamydia	Herpes	Genital warts	Gonorrhea	Syphilis
WOMEN	15.8	7.0	2.7	5.0	4.4	0.5
Race						
White†	15.1	7.3	3.1	5.6	2.8	0.2
Black	19.2	5.5	1.1	2.4	12.6	2.1
Years of education						
≤12	12.1	5.9	1.4	2.8	3.9	1.0
>12	18.8	7.8	3.8	6.8	4.9	0.1
Received public assistance‡						
Yes	23.5	13.0	1.4	5.4	9.1	1.8
No	14.7	6.1	2.9	5.0	3.7	0.3
Income						
< \$10,000	15.1	6.9	1.9	5.3	5.1	1.0
≥\$10,000	16.4	7.1	3.4	4.9	3.9	0.1
MEN	10.2	1.2	0.9	2.5	6.2	0.9
Race						
White	7.8	1.0	1.0	2.6	3.9	0.4
Black	28.0	2.2	0.1	1.3	23.3	3.9
Years of education						
≤12 yrs.	10.0	1.0	0.6	1.7	7.4	0.9
>12 yrs.	10.5	1.4	1.3	3.4	4.8	0.8
Received public assistance						
Yes	14.0	1.3	0.0	1.9	10.6	0.6
No	10.0	1.2	0.9	2.5	6.1	0.9
Income						
<\$10,000	9.9	0.8	0.1	1.0	7.7	1.7
≥\$10,000	10.2	1.3	1.1	2.8	5.8	0.7

*Includes HIV infection, which is not shown separately because of small number of cases. †In this and subsequent tables, "white" includes all racial groups other than black. ‡In this and subsequent tables, refers to assistance received between January 1990 and the survey date.

of differential nonresponse. This weighting permits generalizations from the survey results to the U. S. population represented by the sample.

Data from social surveys are likely to be subject to errors caused by selective non-participation, as well as to measurement error. The interview response rates are respectable for surveys of sexual behavior. Poststratification weighting schemes such as the one employed in these surveys are often used as a partial remedy for errors resulting from selective nonparticipation related to individual characteristics that may be indirectly associated with the dependent variable.³³

Item nonresponse, another possible source of error, is trivial in these two surveys. Nonresponse approaches 2% for income and is 0.1–1.2% for the remaining items. Cases of nonresponse, because of their very small number, have simply been excluded from the analysis.

Variables

The principal outcome variable used in the analysis is a dichotomous measure of whether or not the respondent has ever had an STD. This variable is based on self-reports elicited in a series of questions about AIDS and STDs. Respondents were shown

a flash card with a list of diseases and were asked if they had ever had each one. Subsequently, respondents were asked a series of questions about each disease they reported, including month and year of infection, clinic or private doctor visits for treatment and follow-up, partner notification, and sexual and prophylactic behavior while infected. Individuals were counted as having had an STD if they reported ever having had chlamydia, genital herpes, genital warts, gonorrhea, syphilis or a positive HIV test, or if they had AIDS.

Self-reported history of STD infection is subject to error, most likely underreporting. While some respondents may have deliberately misreported their infection status, others may have had recall problems if the infection was a long time ago and not a serious one. Further, some asymptomatic infections probably were not detected, and thus could not have been reported. Therefore, the estimates obtained from the survey probably represent the lower boundary of the true rates in the population.

While we can make lower boundary estimates and inferences regarding causal relationships with relative confidence, the underreporting of STD infections (deliberate or otherwise) is probably not random and could be related to such factors

as gender, age, race, ethnicity, marital status and socioeconomic status. In particular, if asymptomatic STDs are detected, they generally are detected among infected men and women seeking medical services for unrelated reasons, and these tend to be individuals with the highest levels of use of health care services. The groups with the highest risk of acquiring an STD, however, are typically the least likely to use medical services.³⁴ Consequently, any bias resulting from underreporting of infections is likely to produce attenuated and therefore conservative estimates of the effects of gender, race and socioeconomic status on the likelihood of becoming infected.

As a result of the practical difficulties involved in testing the validity and reliability of self-reported behavioral and medical measures, there are not many sources against which we can check these data. One study concluded that while self-reports of PID in the National Survey of Family Growth probably underestimate overall prevalence, the age and race patterns, and the trends in incidence, agree with information from other databases.³⁵ According to another study, self-reported STD screening in that survey is likely to be an underestimate, yet the data are generally consistent with previous data on risk factors and risk markers for STDs, and the overall quality of the data is good.³⁶ And a recent report concludes that national surveillance data may reflect underreporting of STDs among higher socioeconomic groups using private health care, because private physicians report a small fraction of the STD cases they treat.³⁷ On the other hand, not only are survey data affected by

Table 2. Percentage of respondents who ever had an STD, by background variables

Variable	N	%
Gender		
Male	3,239	10.2**
Female	1,596	15.8
Race		
White	2,908	10.2**
Black	1,927	24.3
Years of education		
≤12 years	2,348	10.7**
>12 years	2,485	13.4
Received public assistance		
Yes	466	20.7**
No	4,315	11.4
Income		
<\$10,000	1,474	12.6
≥\$10,000	3,284	11.9

**Difference within category significant at p<.01. Note: In this and subsequent tables, percentages are based on weighted sample; unweighted Ns are shown.

underreporting of STD experience, but surveys tend to miss persons outside households, who may be at higher risk for STDs.

Finally, aggregation of all infections in a dichotomous dependent variable may conceal subtle patterns of disease acquisition related to differences in knowledge, recognition, symptoms and syndromes, serology and treatment of various infections. Disease-specific analyses would be preferable, but because of the generally low incidence of STDs, a population-based probability survey like the NSM or the NSW is not large enough to identify a sufficient number of infections for such analyses. (We have, however, provided disease-specific rates by race and socioeconomic status measures for men and women in Table 1.) Further, as mentioned earlier, the risk of infection is determined primarily by sexual and health care behavior; there are enough commonalities in the behaviors associated with the acquisition and transmission of these diseases that the effect of pooling them is not likely to detract from the robustness or value of the results.

Our proximate determinants include four types of sexual behavior that are strong risk factors for STD infection: whether or not the respondent has ever had anal intercourse; whether or not the respondent has ever exchanged sex for money or drugs; whether or not the respondent has ever had a onetime sex partner (a "one-night stand"); and the respondent's lifetime number of vaginal sex partners (categorized as one, 2-3, 4-6, 7-15 or 16 or more). Two measures of health care access are included: whether or not the respondent has health insurance, and whether or not the respondent has a regular physician.

The primary exogenous variables of interest are gender, race and class. Race is a dichotomous measure indicating whether the respondent is black or a member of another racial group. (We will refer to the latter category as white, since it contains only small numbers from other racial groups.) The class variable uses three indicators of socioeconomic status: the respondent's educational level (12 or fewer years of schooling vs. more than 12 years); receipt of any public assistance (Aid to Families with Dependent Children, general assistance or food stamps) between January 1990 and the survey date; and personal income (less than \$10,000 vs. \$10,000 or more) in 1990.

In the multivariate analysis, we control for a set of variables related to STD risk, possibly through their effects on sexual behavior and health care access: age; mari-

Table 3. Percentage of respondents who have engaged in various sexual behaviors, percentage distribution by lifetime number of sex partners, and mean number of sex partners, all according to background variables

Variable	Ever had anal sex (N=4,812)	Ever paid for sex (N=4,817)	Ever had one-night stand (N=4,817)	Lifetime no. of partners (N=4,835)						Mean no. of partners
				1	2-3	4-6	7-15	≥16	All	
Gender										
Male	20.8*	6.9**	47.0**	11.6	15.4	18.6	26.5	27.9	100.0**	13.6**
Female	17.9	2.1	26.7	19.1	26.8	25.9	17.3	10.9	100.0	6.7
Race										
White	21.3**	4.8**	41.7**	15.1	18.9	20.7	23.4	21.8	100.0**	11.0**
Black	10.5	8.2	31.7	7.4	20.7	22.8	23.9	25.2	100.0	13.4
Years of education										
≤12	17.8**	5.8*	41.1	12.3	19.7	21.7	23.3	22.9	100.0**	11.6
>12	21.8	4.7	39.6	15.9	18.6	20.2	23.7	21.6	100.0	10.9
Received public assistance										
Yes	22.5	8.7**	42.0	5.5	23.7	26.0	22.6	22.1	100.0**	12.4
No	19.5	5.1	40.1	14.7	18.8	20.8	23.4	22.2	100.0	11.1
Income										
<\$10,000	17.3*	5.7	33.0**	15.1	24.7	22.5	18.1	19.6	100.0**	9.7**
≥\$10,000	20.6	5.2	42.6	13.9	17.2	20.6	25.5	22.9	100.0	11.7

*Difference within category is significant at p<.05. **Difference within category is significant at p<.01; for lifetime number of sexual partners, significance level refers to the difference between distributions. Note: In this table and in Table 4, the N for each column represents the largest number of respondents answering the question; in some categories, Ns were actually 1.5% smaller because of nonresponse.

tal status at the time of the interview; Hispanic origin, based on self-reported ethnicity; and religious affiliation (Catholic, Southern Baptist, other Baptist, conservative Protestant, other Protestant, Jewish or other religion, and no affiliation). We also control for age at first vaginal intercourse because of its strong relationship to subsequent sexual risk-taking behaviors.

Analysis

The analysis is confined to men and women who have ever had sex and is carried out in two stages. First, we conduct bivariate analyses to examine the influence of the exogenous variables (gender, race and class) on the dependent variable (ever had an STD) and on the proximate determinants (health care access and sexual behavior). We then conduct a multivariate analysis, with the controls noted above, to examine the independent effects of gender, race and class on the likelihood of ever having had an STD. Because the dependent variable is a dichotomous outcome, we use a logistic regression approach.³⁸

Three models are estimated. The first includes the measures for gender, race and class, along with the control variables. The second model adds the sexual behavior measures to the first model, and the third model adds the health care access measures to the second.

Bivariate Results

The bivariate relationships between gender, race and class and acquisition of STDs, shown in Table 2, generally support our hypotheses. Women were half again as

likely as men, and blacks were more than twice as likely as whites, to report having had an STD. The class effects vary depending on which socioeconomic status measure is used: A larger proportion of men and women with more than a high school education than of those with less schooling reported having had an STD. Those who received public assistance between January 1990 and the interview date were significantly more likely to report an STD infection than those who did not. The income variable also suggests that higher STD incidence is associated with lower socioeconomic status, but the effect is small and not statistically significant.

We expect STD experience to vary by gender, race and class because of variations in sexual behavior among subgroups. The bivariate associations between sexual behavior and gender, race and class are shown in Table 3. Men were significantly more likely than women to have engaged in sexual behavior that increases the risk of being infected. Blacks were less likely than whites to report anal intercourse and one-night stands, but were more likely to have paid for sex and had a higher lifetime number of sex partners.

The relationship between the socioeconomic status variables and sexual behavior is not as clear-cut. Higher education is associated with an increased likelihood of ever having had anal intercourse, but having paid for sex and having had more than one partner are related to lower education. Reliance on welfare is associated with a higher likelihood of having paid for sex and of having had multiple partners. Anal

Table 4. Percentage of respondents with access to health care, by background variables

Variable	Has insurance (N=4,828)	Has doctor (N=4,834)
Gender		
Male	81.1	58.3**
Female	80.8	75.0
Race		
White	81.2	64.1
Black	79.3	62.2
Years of education		
≤12	74.1**	59.2**
>12	87.7	68.4
Received public assistance		
Yes	69.1**	59.9
No	81.8	64.0
Income		
<\$10,000	62.5**	61.9
≥\$10,000	87.5	64.4

**Difference within category is significant at $p < .01$.

intercourse and one-night stands were more prevalent among the respondents in the higher income group than among those with lower income; lifetime number of sex partners was also higher among respondents whose annual income in 1990 was \$10,000 or above.

Access to health services is strongly associated with various measures of socioeconomic status (Table 4). The proportion of the sample population covered by health insurance increases significantly with education and income, and is higher among respondents who did not receive public assistance in 1990 or 1991 than among those who received some support. Gender and race are unrelated to whether or not the respondent is covered by health insurance. Two characteristics are associated with the likelihood of having a regular physician: being female and having more than 12 years of education.

The conclusion we draw from the bivariate analysis is that sexual behavior and health care access are strongly influenced by race, gender and socioeconomic status, which lead to significant differences in the likelihood of STD acquisition.

Multivariate Results

The first model of the multivariate analysis, which added the five control variables, yields results similar to those of the bivariate analysis. Coefficients and odds ratios are shown in Table 5.

Women were twice as likely as men to report an STD, and blacks were more than twice as likely as whites to do so. Receipt of public assistance and income have no significant relationship to STD experience, but education has a strong association: Men and women with 12 or fewer years

of education were about half as likely as those with more schooling to report an STD infection.

Our data and other studies³⁹ indicate that knowledge of STDs (and therefore the ability to report them) is highly correlated with education. Further, the different STDs varied in the extent to which survey respondents were aware of them. That is, both the level of knowledge and the association of education with awareness varied by disease. For example, the NSM and NSW reveal that knowledge of chlamydia is very strongly related to education, while knowledge of gonorrhea is not. It is also likely that better-educated individuals have greater access to health care services, and therefore are relatively likely to detect an STD infection.

The second model reveals strong positive associations between all four sexual behavior measures and the likelihood of having had an STD infection. Having paid for sex and having had a one-night stand each double the likelihood of infection, and having engaged in anal intercourse raises it by one-half. The odds of having been infected with an STD increase progressively with the lifetime number of sex partners: Compared with those who reported one partner, those who have had 2–3 partners are five times as likely to have had an STD; those with 4–6 lifetime partners, 10 times as likely; and those with 16 or more partners, 31 times as likely.

Including the sexual behavior variables, through which gender, race and class may be expected to operate indirectly, does not reduce the effects of the exogenous variables on the likelihood of an STD infection. In fact, in model 2, the direct effects of gender and race are stronger than these variables' total effects. After the effects of the sexual behavior variables have been accounted for, women and blacks are more than three times as likely as men and whites to have had an STD infection. Further, respondents who have more than a high school education remain significantly more likely than their less-educated counterparts to have reported an STD infection.

According to the results from the third model, neither health care access measure is significantly associated with the likelihood of STD infection; adding these variables to the model therefore does not diminish the effects of the other variables.

We also tested for the effects of two other health care behaviors: ever having used a condom; and having engaged in preventive health care behaviors, including condom and spermicide use or adopting safer sexual practices. Neither of these factors

had significant effects on the likelihood of STD infection, net of the other variables included in the analysis (not shown).

In an additional set of multivariate analyses, we examined whether the relationship between gender and the likelihood of STD infection (through the intervening variables) depends on race, and whether the relationship between race and STD infection is conditioned by gender. That is, we estimated the models in Table 5 separately for men and women, and for blacks and whites. The results (not shown) indicated that the likelihood of STD infection depends on gender among whites, but not among blacks, and depends on race among men, but not among women.

Discussion

Our results from a multivariate analysis of nationally representative data for young adults reveal that the risk of ever having had an STD varies by gender, race and certain aspects of socioeconomic status. We have documented strong relationships between these three variables and sexual and health care behaviors, and between sexual behaviors and the likelihood of STD infection. Yet, direct effects of gender, race and socioeconomic status remain; in fact, the direct effects of gender and race are more pronounced than the total effects.

Several reasons may explain these results. First, the available measures preclude us from estimating models about which we can make strong causal assumptions. Second, the health behavior variables, for which we find no significant effects on the risk of STD infection, measure potential access to health care, but not actual use of health care or timing of use. Third, the lifetime measures of sexual behavior conceal gender, race and class variations in the frequency of high-risk sexual behaviors. Fourth, the outcome measure combines the incidence of several STDs, even though the probability of infection when exposed varies from one disease to another. If there are gender differences in type of infection, then the observed gender effect on lifetime experience with any STD could provide an incomplete picture of the influence of this factor.

Despite these limitations, our study yields some important results. Perhaps most notable is the finding that the direct effect of gender is larger than its effect when measured without the intervening sexual behavior variables. The positive direct effect is larger than the positive total effect because of the negative indirect effect via sexual behavior. Women have fewer sex partners than men and are less

likely than men to engage in risky behaviors. Thus, this finding suggests that if women engaged in risky sexual behavior as much as men did, the rate of STD infection among women would exceed the present level. Likewise, if men modified their sexual behaviors, they would have lower STD rates.

This reasoning also generally applies to the increase in the racial difference in STD infection when the sexual behavior measures are added to the model, although the racial differences in sexual behavior are not as distinct as the gender differences. Blacks and whites alike tend to engage in high-risk sexual behaviors, but in different ones. Nevertheless, when the effects of sexual behavior are held constant, blacks are at an increased risk of STD infection. One possible reason is that the high-risk sexual behaviors in which blacks engage—having multiple partners and paying for sex—expose individuals to a higher risk of STD infection than the high-risk sexual behaviors in which whites engage—one-night stands and anal intercourse. Another possible reason is the differences in rates of infection in black and white respondents' pools of potential partners, which would alter the transmission dynamics within each group.⁴⁰

Data on partner characteristics are available from both the NSM and the NSW, but are reported by the respondents and include partners only during a limited reference period (between January 1990 and survey); hence, they are not very useful for the current analysis. In the future, it would be desirable to have information on racial differences in men and women's pool of potential sex partners and the characteristics of those partners.

With respect to the direct effect of gender, measures related to the biological predisposition to STD infectivity would be desirable. In the adult years, sexual and health behaviors are believed to outweigh biological factors as determinants of the incidence of STD infections, but the contribution of physiological factors cannot be overlooked.⁴¹ Among women, developmental changes in a number of physiological factors—such as the type of lining in the genital tract, the resident flora and acidity of the vagina, and the characteristics of the cervical mucus—affect susceptibility to STDs and their sequelae.⁴²

Physiological changes related to the menstrual cycle, pregnancy and contraceptive use also influence the risk of STD infections. For example, the menstrual cycle appears to influence the risk of upper reproductive tract infection in women, and sexual inter-

Table 5. Logit coefficients and odds ratios showing the likelihood of ever having had an STD

Variable	Model 1 (N=4,600)		Model 2 (N=4,591)		Model 3 (N=4,583)	
	Coefficient	Odds ratio	Coefficient	Odds ratio	Coefficient	Odds ratio
Female	0.72**	2.06	1.24**	3.44	1.24**	3.45
Black	0.86**	2.35	1.18**	3.26	1.16**	3.20
Received public assistance	0.33	1.40	0.07	1.07	0.07	1.08
≤12 yrs. of education	-0.58**	0.56	-0.38**	0.68	-0.36**	0.70
<\$10,000 income	-0.01	1.00	0.07	1.08	0.11	1.12
Ever had anal sex	na	na	0.41**	1.50	0.41**	1.51
Ever paid for sex	na	na	0.67**	1.96	0.68**	1.97
Ever had one-night stand	na	na	0.69**	1.99	0.69**	2.00
Lifetime no. of sex partners						
1	na	na	ref	1.00	ref	1.00
2-3	na	na	1.52**	4.57	1.52**	4.58
4-6	na	na	2.30**	9.98	2.31**	10.04
7-15	na	na	2.87**	17.61	2.87**	17.70
≥16	na	na	3.43**	30.99	3.44**	31.04
Does not have insurance	na	na	na	na	-0.17	0.84
Does not have doctor	na	na	na	na	0.03	1.03
Constant	-2.01		-6.46**		-6.46**	
Log likelihood	-1532.58		-1344.09		-1340.60	
χ ² (df)	295.0 (16)**		662.7 (23)**		660.1 (25)**	

Notes: Models show results net of the effects of age, age at first intercourse, marital status, religion and Hispanic origin; reference categories are not shown for dichotomous variables; na=not applicable. **Difference within category is significant at p<.01.

course during menstruation is associated with a higher STD risk.⁴³ Hygienic practices such as douching and tampon use are considered STD risk markers.⁴⁴ Pregnancy is associated with physiological changes that put women at higher risk of STD infection and its consequences because host defenses are normally suppressed during pregnancy. IUDs and hormonal contraceptives influence the risk of STDs and their sequelae primarily because of their effects on host defenses.⁴⁵ Users of IUDs have been found to run a higher risk of PID than women who are not using contraceptives.⁴⁶ Frequent use of spermicides may cause local inflammation and genital ulceration. Oral contraceptives are thought to increase the risk of cervical chlamydial infections.⁴⁷

In the male, however, little is known about analogous physiological changes that might affect an individual's risk of infection with STDs. Further, seminal and prostatic fluids may contain factors with marked antibacterial activity.⁴⁸ Finally, circumcision appears to reduce the risk of acquiring STDs.⁴⁹

STDs remain a serious problem in the United States and around the world, despite great progress in our understanding of the molecular biology and immunology of the disease mechanisms, and despite the remarkable pace of discovery of effective therapies. Given improvements in the understanding of the pathogenesis and natural history of STDs, and the scientific triumphs in the effective treatment of virtually all nonviral STDs, why have we not been able to control the spread of these diseases?

Our inability to control STDs reflects,

among other factors (such as lack of effective vaccines), the obvious fact that STD prevalence is a function of the sexual and health care behaviors of individuals and groups. These behaviors, which evidently sustain STDs in the population, are directly linked to social, economic and demographic factors. While sexual and health care behaviors can be neither legislated nor controlled in any society, it might be possible to influence them if we knew enough about their determinants and how to effect appropriate behavior modification. Therefore, not only do the social and behavioral factors that contribute to the maintenance of STDs in the population require attention, but such a focus calls for an interdisciplinary approach.

The control and prevention of STDs depends on a more comprehensive understanding of the social and behavioral patterns involved, an ability to identify target populations for behavioral interventions and the capability to design successful interventions. Yet, despite recent national studies, our knowledge of sexual behavior in the general population is rather limited, and research linking sexual and health care behaviors to STD infection typically has relied on ungeneralizable data from clinic populations and convenience samples. Further, while surveillance reports provide useful information about the incidence of a few STDs by age, gender, race and ethnicity, these data are collected at the aggregate level and do not allow micro-level analysis of the effects of predictor variables.

As the results from our study and others illustrate, population-based sample

surveys are useful in producing estimates of complex relationships that may be generalized to the population. Despite the limitations arising from measurement errors related to self-reporting of private behaviors and outcomes, these surveys provide another strong perspective from which STD acquisition and transmission can be viewed. Given the results of our analysis, future research would do well to consider improving the accurate measurement of STD experience and its correlates in an interview setting.

Additionally, such studies should consider including measures that assess the impact of physiological factors, hygienic practices and contraceptive behavior in accounting for the effects of such characteristics as gender, race and socioeconomic status on STD infection. Larger samples would allow disease-specific analysis, which would overcome the problems that result from aggregating diseases in a single outcome measure, and would shed light on relationships that vary by disease and cannot be identified with a combined disease measure. Population-based representative sample surveys, taken together with surveillance reports and clinic-based data, can provide a more complete picture of STDs and social and behavioral factors associated with their acquisition and transmission, and can aid the formulation and implementation of successful interventions.

References

1. Public Health Service, Department of Health and Human Services, *Healthy People 2000: National Health Promotion and Disease Prevention Objectives*, U.S. Government Printing Office, Washington, D.C., 1991.
2. Ibid.
3. Division of STD/HIV Prevention, Centers for Disease Control, *Sexually Transmitted Disease Surveillance, 1990*, Atlanta, 1991.
4. J. E. Anderson, L. McCormick and R. Fichtner, "Factors Associated with Self-Reported STDs: Data from a National Survey," *Sexually Transmitted Diseases*, **21**: 303-308, 1994.
5. A. P. Polednak, *Racial and Ethnic Differences in Disease*, Oxford University Press, New York, 1989.
6. A. A. Ehrhardt and J. N. Wasserheit, "Age, Gender, and Sexual Risk Behaviors for Sexually Transmitted Diseases in the United States," in J. N. Wasserheit, S. O. Aral and K. K. Holmes, eds., *Research Issues in Human Behavior and Sexually Transmitted Diseases in the AIDS Era*, American Society for Microbiology, Washington, D. C., 1991, pp. 97-121; H. Amaro and I. Gornemann, "Health Care Utilization for Sexually Transmitted Diseases: Influence of Patient and Provider Characteristics," *ibid.*, pp. 140-160; and R. B. Jones and J. N. Wasserheit, "Introduction to the Biology and Natural History of Sexually Transmitted Diseases," *ibid.*, pp. 11-37.
7. N. S. Padian, S. C. Shiboski and P. J. Hitchcock, "Risk Factors for Acquisition of Sexually Transmitted Diseases

and Development of Complications," in J. N. Wasserheit, S. O. Aral and K. K. Holmes, 1991, op. cit. (see reference 6), pp. 83-96.

8. S. O. Aral et al., "Demographic and Societal Factors Influencing Risk Behaviors," in J. N. Wasserheit, S. O. Aral and K. K. Holmes, 1991, op. cit. (see reference 6), pp. 161-176; and A. E. Washington et al., "Assessing Risk of Pelvic Inflammatory Disease and Its Sequelae," *Journal of the American Medical Association*, **266**:2581-2586, 1991.
9. S. L. Hofferth, "Factors Affecting Initiation of Sexual Intercourse," in S. L. Hofferth and C. D. Hayes, eds., *Risking the Future: Adolescent Sexuality, Pregnancy, and Childbearing*, Vol. 2, National Academy Press, Washington, D. C., 1987, pp. 7-35.
10. J. O. G. Billy et al., "Sexual Behavior of Men in the United States," *Family Planning Perspectives*, **25**:52-60, 1993.
11. H. Amaro and I. Gornemann, 1991, op. cit. (see reference 6).
12. I. L. Reiss, *Journey into Sexuality: An Exploratory Voyage*, Prentice-Hall, Englewood Cliffs, N. J., 1986; and ———, "A Sociological Journey into Sexuality," *Journal of Marriage and the Family*, **48**:233-242, 1986.
13. G. S. Becker, *A Treatise on the Family*, Harvard University Press, Cambridge, Mass., 1981.
14. H. Amaro and I. Gornemann, 1991, op. cit. (see reference 6).
15. G. S. Becker, 1981, op. cit. (see reference 13).
16. A. A. Ehrhardt and J. N. Wasserheit, 1991, op. cit. (see reference 6).
17. Ibid.
18. Ibid.
19. J. D. Forrest and S. Singh, "The Sexual and Reproductive Behavior of American Women," *Family Planning Perspectives*, **22**:206-214, 1990.
20. E. O. Laumann et al., *The Social Organization of Sexuality: Sexual Practices in the United States*, University of Chicago Press, Chicago, 1994.
21. S. O. Aral et al., "Sex Partner Selection as Risk Factor for STD: Clustering of Risky Modes," *Sexually Transmitted Diseases*, **18**:10-17, 1991.
22. J. H. Hibbard and C. R. Pope, "Gender Roles, Illness Orientation, and Use of Medical Services," *Social Science and Medicine*, **17**:129-137, 1983; and H. W. Neighbors and C. S. Howard, "Sex Differences in Professional Help Seeking Among Adult Black Americans," *American Journal of Community Psychology*, **15**:403-417, 1987.
23. A. A. Ehrhardt and J. N. Wasserheit, 1991, op. cit. (see reference 6).
24. Ibid.
25. J. O. G. Billy et al., 1993, op. cit. (see reference 10).
26. K. Tanfer et al., "Condom Use Among U. S. Men, 1991," *Family Planning Perspectives*, **25**:61-66, 1993.
27. R. Anderson and J. P. Newman, "Societal and Individual Determinants of Medical Care Utilization in the U. S.," *Milbank Memorial Fund Quarterly*, **51**:95-124, 1973.
28. E. O. Laumann et al., 1994, op. cit. (see reference 20).
29. S. O. Aral et al., 1991, op. cit. (see reference 8).
30. E. O. Laumann et al., 1994, op. cit. (see reference 20).
31. K. Tanfer, "National Survey of Men: Design and Execution," *Family Planning Perspectives*, **25**:83-86, 1993.
32. ———, "National Survey of Women: Design and Execution," Battelle Memorial Institute, Seattle, 1995.
33. M. J. Banks, "Weighting the Data," in R. Anderson, J. Kasper and M. R. Frankel, eds., *Total Survey Error: Ap-*

plications to Improve Health Surveys, Jossey-Bass, San Francisco, 1979, pp. 104-112; and M. J. Banks and M. R. Frankel, "Adjusting for Total Nonresponse," *ibid.*, pp. 87-96.

34. L. A. Aday and S. M. Shortell, "Indicators and Predictors of Health Services Utilization," in S. J. Williams and P. R. Torrens, eds., *Introduction to Health Services*, Delmar Publishers, Albany, N. Y., 1988, pp. 51-81.
35. S. O. Aral, W. D. Mosher and W. Cates, "Self-Reported Pelvic Inflammatory Disease in the United States," *Journal of the American Medical Association*, **266**:2570-2573, 1991.
36. W. D. Mosher and S. O. Aral, "Testing for Sexually Transmitted Diseases Among Women of Reproductive Age: United States, 1988," *Family Planning Perspectives*, **23**:216-222, 1991.
37. J. E. Anderson, L. McCormick and R. Fichtner, 1994, op. cit. (see reference 4).
38. E. A. Hanushek and J. E. Jackson, *Statistical Methods for Social Scientists*, Academic Press, New York, 1977.
39. J. E. Anderson, L. McCormick and R. Fichtner, 1994, op. cit. (see reference 4).
40. R. M. Anderson, "The Transmission Dynamics of Sexually Transmitted Diseases: The Behavioral Component," in J. N. Wasserheit, S. O. Aral and K. K. Holmes, 1991, op. cit. (see reference 6), pp. 38-60.
41. A. A. Ehrhardt and J. N. Wasserheit, 1991, op. cit. (see reference 6).
42. Ibid.
43. K. Tanfer and S. O. Aral, "Sexual Intercourse During Menstruation Among Young Sexually Active Women, United States, 1991," Seattle, 1995.
44. N. S. Padian, S. C. Shiboski and P. J. Hitchcock, "Risk Factors for Acquisition of Sexually Transmitted Diseases and Development of Complications," in J. N. Wasserheit, S. O. Aral and K. K. Holmes, 1991, op. cit. (see reference 6), pp. 83-96.
45. D. A. Grimes and W. Cates, Jr., "Family Planning and Sexually Transmitted Diseases," in K. K. Holmes et al., eds., *Sexually Transmitted Diseases*, McGraw-Hill, New York, 1990, pp. 1087-1094.
46. D. A. Grimes, "Intrauterine Devices and Pelvic Inflammatory Disease: Recent Developments," *Contraception*, **36**:97-109, 1987.
47. A. E. Washington et al., "Oral Contraceptives, Chlamydia Trachomatis Infection and Pelvic Inflammatory Disease," *Journal of the American Medical Association*, **253**:2246-2250, 1985.
48. A. A. Ehrhardt and J. N. Wasserheit, 1991, op. cit. (see reference 6).
49. S. O. Aral and K. K. Holmes, "Epidemiology of Sexual Behavior and Sexually Transmitted Diseases," in K. K. Holmes et al., 1990, op. cit. (see reference 45), pp. 19-36.